

Stanford type-A an aortic dissection onset with neurological symptoms: a challenging case to diagnose

Aortic dissection with neurological symptoms

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Abstract

A 71-year-old male patient with a sudden onset of ambiguity, difficulty in recognizing people, difficulty speaking, and backache was admitted to the emergency service. First radiological imaging was routine. Second imaging revealed acute stroke, but these complaints were considered not to be related to the stroke. He underwent thorax computerized tomography angiography because of the suspicion of aortic dissection (AD) and was interpreted as Stanford Type-A AD. The onset of AD with neurological manifestations is very rare. Clinicians should be alert for AD in cases of unusual combinations of nervous system symptoms and/or concomitant syncope, seizures, cerebral, spinal, peripheral ischemia.

Keywords

Chest Pain; Stroke; Aortic Dissection; Neurological Symptoms

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Introduction

Aortic dissection (AD) consists of the separation of the layers of the aortic wall [1]. AD, the incidence of which ranges from 5 to 30 million, generally manifests with acute thoracic/abdominal pain and accompanied by cardiovascular manifestations. Clinical symptoms are wide and highly variable. Acute onset neurological symptoms in AD do not show any serious pain in one-third of the patients [2,3]. Neurological manifestations are caused by hypotension, occlusion of the cerebral vessels, and perfusion deficit. [1,4]. Here, we presented a rare case with Stanford Type-A Aortic Dissection (STAAD) that started with neurological symptoms.

Case Report

A 71-year-old male patient with a sudden onset of ambiguity, difficulty in recognizing people, difficulty speaking, and backache was admitted to the emergency service. In the patient's anamnesis, it was learned from himself and his relatives through nonverbal communication that there was a growing pain in her back and chest after her complaints of difficulty in recognizing and speaking. At the time of admission, the overall situation was moderate, confused, and agitated. He has had a story of hypertension for 23 years and 56 packs/ year of smoking cigarettes. Apart from these, there were no chronic illnesses and no operative stories. Vital findings were as follows: blood pressure arterial (BPA) recorded in the right arm was 80/40 mmHg, but it could not be obtained from the left side; temperature: 36.7 Centigrade, pulse: 62 beats / min; saturation: 96%; respiratory rate: 12; blood sugar: 106 mg/dl. Physical examination was normal except the bilateral, lower extremities were cold and cyanotic. Electrocardiography showed no ischemic signs. Immediately after the general condition of the patient was stabilized, radiological imaging was performed with an acute stroke pre-diagnosis. No hemorrhage area or hypodense was detected in computerized brain tomography (CT). Diffusion magnetic resonance imaging (MRI) revealed acute stroke (Figure 1), but these complaints were considered not to be related to the stroke. The anamnesis and physical examination suggested AD that we were strongly suspected. The patient underwent contrast-enhanced thorax CT angiography (Figure 2) and was interpreted as STAAD in the aortic arch (Figure 3). The patient underwent an emergency operation, including artificial aorta and semi arc replacement with synthetic graft. The patient was lost on post-operative day two due to ventricular fibrillation. Written informed consent was obtained from the patient's legal custodian for publishing the individual medical records.

Discussion

AD risk factors in the literature have been well designated. We know very little about pathological and molecular events before or after AD because of the sudden and unpredictable nature of AD. In a recent study in line with the findings of human AD, it was determined that focal medial deterioration occurred with inflammatory and proliferative response activated due to ongoing aortic stress [2]. Acute AD is defined as 14 days after onset of symptoms, subacute between two weeks and two months, and chronic dissection after two months. AD is

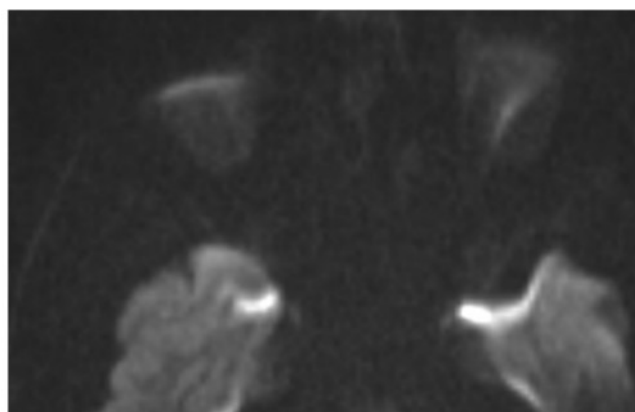


Figure 1. A) Millimetric hyperintense in the right cerebellar hemisphere in the diffusion-weighted images, and B) acute ischemia is seen in the ADC field in this area (arrowheads).

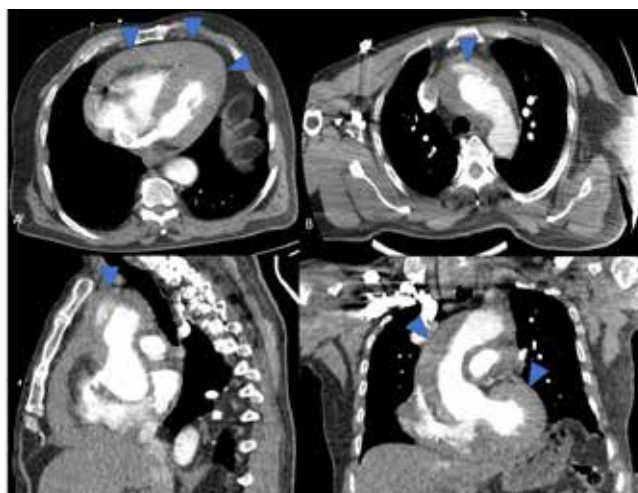


Figure 2. A) Millimetric hyperintense in the right cerebellar hemisphere in the diffusion-weighted images, and B) acute ischemia is seen in the ADC field in this area (arrowheads).



Figure 3. Diameter increase is observed in the ascending aorta and aortic arch in the 3D volumetric rendering images obtained from Contrast-enhanced thorax CT 3D angiography. A) Right and B) left oblique sagittal images depict the morphologic features and initial of the STAAD, C) coronal image of the STAAD with bones, D) semi-oblique sagittal image shows enhanced hemopericardium

categorized using two separate systems: the first of these is the Stanford classification. Type A is called the involvement of only the ascending aorta or co-involvement of both ascending and descending aorta and type B is called dissection of the aorta distal to the subclavian artery.

Although the generally sudden onset of severe chest-back pain and hypotension most commonly associated with acute myocardial infarction are the most common symptoms, clinical manifestations are comprehensive and highly variable depending on the location of AD. However, 10-55% of the cases reported painless AD. Neurological symptoms occur due to the occlusion of the carotid, vertebral, spinal arteries, and peripheral nerves as a result of the occlusion of the vessels or due to the decrease in cerebral perfusion due to hypotension. In the literature, there is a small amount of data about neurological symptoms of AD patients. Stroke was observed in 21 patients with AD, and increased mortality and morbidity were also seen in the hospital, but not with long-term mortality rate [4].

AD-associated with neurological symptoms is only 17- 40%, but it is dramatic and may mask the underlying cause [1]. In our case, the first symptom was the speech difficulty that developed following the failure to recognize the environment. For this reason, after the general condition of the patient has been improved, the first desired examination was brain CT. No acute pathology was considered. Subsequently, despite the detection of acute ischemia on the MRI, the patient underwent contrast-enhanced thoracic CT angiography and was interpreted as STAAD in the aortic arch. (Figure 2,3).

Although the latest developments in modern diagnostic methods, surgical treatments, and medical devices, it still has high mortality. When not treated, 75% of ADs, especially with STAADs, are lost within two weeks. Emergency surgery is now the only option to rescue STAAD patients. However, surgical mortality has remained unchanged at 10 to 35%, even in experienced centers. Moreover, long-term survival results are poor [2]. No matter how they are treated, aortic rupture among all the causes of mortality in STAAD is the most dangerous and feared one. One-third of the deaths reported in patients who are not surgically repaired or treated conservatively are STAAD, and it can rapidly lead to fatal conditions such as aortic rupture and multiple organ malperfusion.

It has been proven that imaging methods help diagnose STAAD. Despite everything, less than half of AD has been diagnosed correctly. Although they are available in every emergency service, it is time-consuming or not appropriate for hemodynamically unstable patients. The selection of necessary diagnostic tests may be critical for the survival of these patients since the development of complications is time-dependent in the future [5,6]. In our case, no radiological examinations were performed until the patient's condition stabilized.

In conclusion, the correct diagnosis may be difficult in emergency departments due to the variety of symptoms and resemblance to other diseases. To improve clinical outcomes, it is necessary to develop strategies that will predict the onset of AD and prevent the emergence of more critical things to avoid natural progression. Here, the essence of diagnosis is high suspicion in the patient with atypical symptoms.

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and human rights statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article.

Conflict of interest

None of the authors received any type of financial support that could be considered potential conflict of interest regarding the manuscript or its submission.

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